ATTACHMENT 2

A REVIEW OF CURRENT EXPOSURES AND CURRENT HEALTH RISKS ASSOCIATED WITH ASBESTOS EXPOSURE IN LIBBY, MONTANA

REPORT BY:
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QUALIFICATIONS

1. I am President and Chief Executive Officer of Sciences International, Inc., a company of scientists dedicated to health and environmental assessment.

2. I have a Ph.D. in organic chemistry and am a Fellow of the Academy of Toxicological Sciences. Formerly, I spent 14 years at the U.S. Environmental Protection Agency (EPA), from October, 1971 to December 1985, where I directed EPA's central risk assessment programs for the last 10 years of my tenure. Specifically, in 1975, I became the Executive Director of an intra-Agency committee that was commissioned to write an Agency cancer policy. This committee developed the Agency's first risk assessment guidelines for assessing risk associated with exposure to suspected carcinogens in the environment. Subsequently, in 1976, I established EPA's first Carcinogen Assessment Group (CAG) which formed the core for the enlarged office, the Office of Health and Environmental Assessment (OHEA), now called the National Center for Environmental Assessment, which was established in 1978. As the director of the first CAG and then OHEA, I had responsibility for the central risk assessment activities of EPA for 10 years before I left the Agency. The primary functions of this office were to conduct assessments and establish the toxicity of a wide variety of toxic agents, provide leadership to establish EPA-wide guidelines for toxicity and risk assessments, and oversee EPA's health assessment programs. Of particular relevance to the issues herein addressed, my office was responsible for the risk assessment of toxic air pollutants and for writing the National Ambient Air Quality Criteria Documents. Among the scientific documents for which I was responsible during this term was the draft document, Airborne Asbestos Health Assessment Update (EPA, 1986a), which was published in June 1986, shortly after my departure.

3. Since leaving EPA, I have continued active participation in the sciences of health and environmental risk assessment. For example, I am past-President of the Society for Risk Analysis; and am currently Editor-in-Chief of the journal, *Risk Analysis: An International Journal*, which is the leading peer-reviewed international journal on topics of risk assessment. I regularly serve on expert peer review and advisory committees on risk assessment topics for the EPA and other organizations including current appointments to the Department of Energy's Los Alamos National Laboratory Expert Advisory Committee and a recent expert committee for the National Academy of Sciences. My curricula vitae including a list of my publications for the last ten years is provided in Appendix A. A list of recent depositions and trial testimony is provided in Appendix B. My compensation is provided in Appendix C.

4. I am submitting this report in support of WR Grace's defense in the cost recovery case related to alleged asbestos contamination in Libby, Montana.

5. I have reviewed the risk assessments prepared by Chris Weis of EPA Region VIII, the Action Memoranda for the Libby site, and the associated administrative record. On the basis of my review of this information, I am submitting this report on behalf of WR Grace as evidence of significant scientific deficiencies in the characterization of health risks to Libby residents from current (or pre-remedial) asbestos exposures. These deficiencies are detailed in the following sections.

I. The time-critical removal at Libby that EPA conducted should have been motivated by current exposures (or pre-remedial)[1] and related risks. However, EPA presents numerous analyses and studies that relate to historical exposures that were much higher. The most relevant analysis that EPA has presented is the Weis screening level risk assessments, which are the focus of this report.

A. Risk assessment is the scientifically accepted method for assessing the potential health effects associated with current exposures that might be associated with future health risks.

I have been involved in the assessment of risk from environmental contaminants since the 1970s. The accepted method for analyzing the potential effects from current exposures that might be associated with health effects later in life, such as cancer, is risk assessment. These methods and approaches have been widely adopted by state, national, and international organizations as an appropriate basis for making health policy decisions. EPA has provided numerous analyses and assessments, but the most relevant analyses are the health risk assessments by Weis.

B. There is a long latency period for asbestos-related diseases. Therefore, illnesses observed today are likely the result of high-level exposures that occurred before the mining and related operations were closed. Therefore, earlier, high-level exposures do not justify the current time-critical removal. This distinction has not been clearly made by EPA.

Asbestos diseases have long latency periods, meaning that the disease does not become apparent until long after the exposure occurs. The approximate latency periods are as follows: (1) asbestosis: 25-40 years, (2) mesothelioma: 15-30 years or more, and (3) lung cancer: 15-30 years[2]. Therefore, any observed illnesses today in Libby are likely due to historical exposures. ATSDR recognized this fact in its mortality study that focused on deaths occurring between 1979 and 1998, because "the highest exposures to asbestos in Libby are thought to have occurred from the 1950s through the early 1970s." Likewise, the ATSDR medical monitoring study only included subjects who were exposed prior to the closure of the mining operations, under the assumption that exposures were highest during this time period. Both the Weis screening level risk assessments and EPA's related action memoranda reference these studies without proper consideration of the timing of exposure, level of exposure, or duration of exposure, all of which differ from current conditions.

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[1] For the remainder of the document, "current" exposures will refer to both the current situation in Libby and the pre-remedial situation. Pre-remedial refers to the 1999 and subsequent removal actions. In both cases, the term is used to differentiate between exposures after and prior to the closure of the mining operations in 1990.

[2] EPA document entitled "What is asbestos," available at http://www.epa.gov/opptintr/asbestos/asbe.pdf .

C. *The mining operations ceased in 1990, which removed the primary source of asbestos and reduced occupational and residential exposures. Also, other changes at the mining operations prior to 1990 reduced exposures compared to prior periods. The distinctions made in the Weis screening level risk assessment documents and the EPA action memoranda are about occupational and non-occupational exposure and risk. The relevant delineation should not be occupational versus non-occupational, but rather should regard the level, duration, and frequency of exposure.*

The mining operations in Libby ceased in 1990. Therefore, prior to the closure of the mine, there was a potential for high exposures due to the quantities of materials being processed at the mining and milling sites. There was ample opportunity for exposures to asbestos between the 1940s through 1990, with larger exposures in the earlier years. There were several operations in or near town during this period: (1) the mine and mill on Rainy Creek Road on top of Zonolite Mountain, (2) the screening plant and railroad loading station at the intersection of Rainy Creek Road and Highway 37, (3) the expansion/export plant located off of Highway 37, and (4) the expansion plant in the town of Libby (went offline sometime in the 1950s). Before the mine closed, Libby produced 80% of the world's supply of vermiculite.

There is no disagreement that prior levels (before 1991) of asbestos exposure to the Libby population were significantly higher than current exposure levels. There is strong evidence that changes in the milling process and attendant emission control equipment over time reduced the potential for high exposures, both for occupational and non-occupational settings. Once operations ceased in 1990, the potential for prolonged high dose non-occupational exposures was significantly reduced. This is evidenced by the lack of significant asbestos ambient air concentrations in the Libby vicinity for the sampling conducted by EPA after the mine closure in 1990.

A study conducted by Amandus et al. (1987a) supports the assumption that the highest exposures would have occurred during operations prior to the early 1970's. The Amandus study developed exposure estimates for workers at the vermiculite mining and milling sites up to approximately 1982. Their analysis was based on measured historical air concentrations collected by the State of Montana, various federal agencies (NIOSH, MESA, and MSHA), the Zonolite Company, and W.R. Grace. Amandus concluded that the exposure levels decreased significantly after approximately 1974. The highest exposure levels predicted by Amandus were for the Dry Mill jobs where estimates of exposure prior to 1964 ranged from 168 f/cc in the working areas to 182 f/cc for sweepers. Amandus estimated that the exposures for the same areas during the period from 1964 to 1971 were 33 f/cc and 36 f/cc, respectively. This decrease in exposures levels is attributable to changes in operations and control equipment at the facility. A new wet mill operation went into operation in 1974, replacing the dry

mill. Amandus estimated that exposure levels in the new wet mill were 3.2 f/cc in 1974, and 0.8 f/cc from 1977 to the end of the study period in 1982.

EPA also has noted that the potential for high dose exposures to asbestos for the Libby population (i.e., non-occupational exposure) has substantially decreased since the mine closure in 1990. In the screening level risk assessment conducted for Libby residents exposed to ambient air, EPA defines two exposure periods, one to represent mine and mill operations prior to 1990, and a second that represents exposures post 1990, after the mine and mill had closed (Weis, April 2002 memo). The exposure point concentration for the post 1990 period is 1,000 times lower than the value for periods when the mine and mill were operating[3].

EPA states the following in its first action memorandum "Descriptions of historic operations of the mine, mill, and processing centers indicated that large amounts of dust and other fugitive emissions were released into the environment when these operations were still running."

D. *In presenting the basis for the time-critical removal action at Libby, EPA has included a variety of studies and analyses, which are not relevant because the documents do not address current exposures.*

The studies and analyses put forth by EPA include:

- Epidemiologic studies demonstrating that workers associated with the Libby mining operations had elevated levels of disease.
- A mortality study showing that the Libby population has elevated levels of asbestos-related disease.
- A study showing that Libby has an elevated incidence of pleural plaques (lung abnormalities) and the incidences are related to pathways of exposure that do not exist any longer (see analysis of this study in the next section).
- Reports from a pulmonologist (Dr. Whitehouse) that he is treating several non-occupational cases of asbestos related disease.
- Screening level risk assessments based on measurements of exposure made by EPA in Libby and the EPA cancer potency factor for asbestos.

The only directly relevant analysis are the screening level risk assessments because they are the only analysis that address current risks. Workers in the mining operations in Libby clearly had elevated levels of asbestos related diseases. It is also possible that elevated, historical non-occupational exposures may have caused some asbestos related disease, and could have resulted in the elevated incidence of pleural plaques among Libby residents. EPA provides some

[3] The pre-1990 exposure point concentration used by Weis of 1 fiber/cc may not be representative. Amandus reported three measurements in a "downtown office building" in the 1970s. Two measurements were zero (but possibly with less sensitive detection than today), and the other measurement was 0.2 fibers/cc.

evidence to support the relationship, but this evidence is poorly documented and does not meet the minimum requirements for scientific rigor. Nevertheless, the differentiation that EPA makes between occupational and non-occupational exposure is not the relevant issue. The relevant issue is the difference between high and low exposures. The historical exposures in Libby, whether occupational or non-occupational, were substantially higher than exposures in Libby today. Current remediation of the site at Libby must be motivated by the risk of current exposures and associated risks, not historical exposures.

E. *The most relevant piece of evidence provided by EPA for the removal action is the screening level risk assessments. A health risk assessment is the scientifically accepted method for addressing the potential risk of current exposures. However, there are deficiencies in the Weis screening level risk assessments, which are discussed below.*

The relevant analysis presented by EPA for its actions are the screening level risk assessments by Weis. Most of the other studies relate to historical exposures that were much higher than today. The remediation of the site should only be based on current exposures and associated risks. However, the screening level risk assessments presented by EPA, which address current exposures, have substantial scientific deficiencies, which are discussed in this report. A more careful analysis of the monitoring data collected by EPA has been completed by RJ Lee (Lee, 2002). This report presents a corrected risk assessment based on these data.

II. The Weis risk assessments and EPA action memoranda present a variety of analyses of asbestos related health effects in Libby, but do not distinguish prior exposure and risk from current exposure and risk, which is clearly lower. It is not scientifically supportable to use these earlier historical studies to describe current exposure and risk. The available health effects data in Libby are consistent with occupational exposures and likely non-occupational exposures prior to the closure of the mine and prior remedial activities in Libby. It is misleading and scientifically indefensible to attribute these risks to current conditions in Libby.

A. The Weis risk assessments and EPA action memoranda present epidemiologic studies showing that occupational exposure to Libby fibers resulted in asbestos-related diseases.

EPA cites the studies of Lockey et al. (1984), Amandus et al. (1987b), and McDonald et al. (1986), which show elevated levels of asbestos related disease associated with occupational exposures to Libby fibers. EPA erroneously presents these studies as evidence of current risk associated with Libby fibers.

There is no question that occupational exposure to asbestos, including Libby amphiboles, has resulted in elevated incidences of asbestos related diseases. However, the exposures in these studies were substantially higher than exposures in Libby today. For example, Amandus estimated that exposure point concentration for the workers ranged from 1 to 182 fibers/cc, depending upon the type of job and the time period (the implementation of engineering controls and other changes reduced exposures over time). The exposure point concentrations in Libby today are much lower (see later discussion). Therefore, the occupational studies address substantially higher exposures than exist at Libby today.

B. The ATSDR mortality study showed that there were 11 cases of asbestosis in Libby, which ATSDR states is between 40-60 times the national average incidence. EPA uses this study as evidence that its removal action is necessary, but the study is not relevant to current exposures.

The ATSDR mortality study examined death certificates between 1979 and 1998. Given the latency period for asbestos related illnesses, the relevant exposure period for these illnesses would be well before the closure of the mining operations in 1990. Therefore, the exposures in question would be substantially higher than those seen in Libby today.

Furthermore, the mortality study did not address whether the illnesses were from occupational or non-occupational exposures. Given that 10 of the 11 cases of asbestosis were in males, occupational exposures, where levels were reportedly high, are likely for at least most of the cases. Also, given that many people in Libby, a relatively small town, were involved in the mining operations prior to 1990, it is not surprising that Libby has a high rate of asbestos related disease

compared to other areas without asbestos-related industries. However, this high rate of disease from historical exposures does not mean that current, lower exposures present a compelling risk.

C. *The ATSDR medical screening study correlates a variety of exposure scenarios with pleural plaques, but none of the exposure scenarios that exist today were correlated with pleural plaques.*

In the ATSDR medical screening study, the presence of pleural plaques was correlated with various exposure pathways obtained from an interview with the study participants. The study included people "if they were former workers of WR Grace/Zonolite Company (WRG), secondary contractors of WRG, household contacts of former WRG workers, or had resided, worked, attended school, or participated in activities in the Libby area for a period of 6 or more months before December 31, 1990." Therefore, the focus of the study is on exposures prior to the closure of the mining operations.

EPA asked each study participant if they ever engaged in a particular exposure pathway (e.g., played in vermiculite piles, used vermiculite for gardening, etc.). These responses were correlated with pleural plaques using a multivariate regression analysis.

Several exposure pathways were found to be associated with pleural abnormalities: (1) ever worked at Grace or Zonolite, (2) secondary contractor work, (3) played at baseball field near the expansion plant (only marginally statistically significant), (4) played in vermiculite piles, (5) popped vermiculite, and (6) lived with Grace or Zonolite workers. The largest factors were nos. 1 and 6. Most of these pathways are no longer possible. Any occupational related pathway (nos. 1, 2 and 6) no longer exists. The baseball field no longer exists (no. 3), and residents are no longer bringing home vermiculite from the mine where it might be "popped" (no. 5). The only possible remaining pathway might involve small vermiculite piles; however, it is my understanding that most vermiculite piles were removed by the time the mine closed. Therefore, the findings in this study do not provide significant evidence that current exposures are related to lung abnormalities.

In addition, some of the exposure scenarios that may exist today or prior to remediation were not associated with pleural abnormalities, including: (1) vermiculite insulation in homes, (2) used vermiculite for gardening, (3) used vermiculite around the home, (4) handled vermiculite insulation, and (5) recreational activities along Rainy Creek road. Also, the ATSDR results do not support EPA's action at Rainy Creek road.

D. *EPA mentions reports from a Spokane pulmonologist that he is treating several cases of asbestos-related disease in individuals with an occupational and possible non-occupational exposure history. However, there is very little documentation available to verify the reports from Dr. Whitehouse. Nevertheless, any of the illnesses among the patients of Dr. Whitehouse are likely due to historical exposures, not current exposures, given the latency of asbestos related disease.*

This issue is addressed in the report being prepared by Dr. Suresh Moolgavkar. Dr. Moolgavkar, a noted biostatiscian and epidemiologist, has found that the documentation provided by Dr. Whitehouse was inadequate to conduct a review of the conclusion that some cases were not related to occupational exposures, which seems to imply a particular relevance to current Libby exposures (Moolgavkar, 2002). As discussed alone, this implied relevance is inappropriate because of the evidence that exposures were considerably higher prior to 1991. Nonetheless, given the latency period for asbestos-related illnesses, the illnesses of his patients are likely the result of historical exposures, not current exposures.

E. *A presentation by Dr. Marcel Goldberg is cited by EPA as evidence that non-occupational exposures to asbestos are associated with asbestos-related diseases. However, the studies cited by Goldberg refer to significantly higher exposures than currently exist in Libby.*

The presentation by Dr. Goldberg at the Asbestos Health Effects Conference in Oakland, California, in May of 2001, discussed numerous epidemiological studies from sites around the world where environmental exposures to amphibole asbestos were known to occur. Dr. Goldberg concludes that there is "evidence of strong effects of non-occupational exposures." He also notes that the intensity of the exposure is a key determinant of risk to the exposed individual. In other words, Dr. Goldberg concluded that the exposure level, or concentration of airborne asbestos is a major factor in determining the potential for an adverse health effect. Other factors cited by Dr. Goldberg as critical to the occurrence of disease from asbestos exposures are related to the frequency and duration of the exposures.

The exposure pathways cited by Goldberg are substantially different from the situation in Libby. The non-occupational exposures described by Dr. Goldberg reflect exposures to high concentrations of airborne asbestos, or they are associated with frequent exposure to individuals with occupational asbestos exposures. The occurrence of asbestos disease in individuals whose only exposure is via another individual who has occupational exposures is only relevant to conditions in Libby prior to the mine closing in 1990. Other non-occupational exposures described by Dr. Goldberg include individuals who quarried and ground rocks containing very high levels of tremolite asbestos to create a whitewash that was then applied to homes in Greece and Turkey (Yazicioglu, 1980, Dumortier, 2001). In addition, in New Caledonia, non-occupational asbestos exposure levels have been reported as high as 0.67 f/cc for

areas near tremolite mines, and 78 f/cc for sweeping houses (Bourdes, 2000). Clearly, these levels do not reflect the current exposure levels for the population of Libby post-1990.

F. *EPA erroneously equates exposures observed in the Marysville, Ohio epidemiology study to potential exposures at the screening and export plants. A careful review of this claim shows that EPA's analysis is incorrect for several reasons.*

Lockey et al. (1984) investigated pulmonary changes after exposure to "mainly Montana" vermiculite in a group in Marysville, Ohio workers. EPA claims that the "medium" exposure group, which "consisted of employees who worked in the warehouse, in packaging and in central maintenance" and had "low level fiber exposure," had an elevated level of "lung abnormalities" (EPA comments to Grace, June 4, 2002). EPA then states that the exposure in this "medium" exposure group was similar to the current exposures in the screening and export plants, so one could expect "lung abnormalities" for residents or workers at the screening and export plant (prior to the cleanup). EPA's comments are incorrect for three reasons:

1) The "medium" exposure group in Marysville was not associated with "lung abnormalities." EPA misread the results of the Lockey study.
2) The exposures at the screening and export plants are lower than stated by EPA. EPA made a calculation error that accounts for their incorrect statement associating the screening and export plant exposures with the medium exposure group in the Lockey study.
3) The exposures at the screening and export plants as reported from EPA's measurement data were significantly lower than the exposures that Lockey found associated with lung abnormalities, and were even lower than the control group in the Lockey study.

EPA states on page 5 of its comments to Grace:

> "The medium group [in the Lockey study] consisted of employees who worked in the warehouse, in packaging, and in central maintenance. This group had a 5% prevalence of lung abnormalities (Table #4, Page 955), twice that of the low exposure group and half that of the high exposure group. Thus, the study confirmed a exposure-response relationship between asbestos exposure and lung abnormalities."

EPA has misread the Lockey results. The results in Table 4 of the Lockey study do not refer to the comparisons between the "low," "medium," and "high" exposure groups. In addition to these exposure classifications, Lockey also classified the subjects by their individual cumulative exposures that account for where they worked and how long they were employed, which is what is referred to in Table 4 of his paper. The first classification (low, medium, and high) refers

only to job classifications, while the cumulative exposure classification accounts for both job classification and the number of years employed. In this table, Lockey reports that subjects with cumulative exposures between 1-10 fibers/ml-year had a 5% prevalence of lung abnormalities, compared to 2.4% in a "control" group with less than 1 fiber/ml-year cumulative exposure. The difference between these two groups is not statistically significant by Fisher's Exact Test ($p=0.11$)[4], but relatively close to statistical significance. The results for the three exposure groups (low, medium, and high) are presented in Table 5, not Table 4. For the medium exposure group, there was a 3.9% prevalence of lung abnormalities, while there was a 2.8% prevalence in the low exposure group. This small change is not statistically significant by Fisher's Exact Test ($P=0.44$), showing that the medium exposure group is not associated with a statistically significant elevated level of lung abnormalities. Furthermore, the lung abnormalities were defined by Lockey as either costrophrenic angle blunting only or pleural/parenchymal changes. The low exposure group actually had a higher incidence than the medium exposure group for pleural/parenchymal changes (1.9% for low group and 1.5% for medium group), which was the most significant effect tested in the study according to Lockey. The average cumulative exposures for the medium group were about 1 fiber/ml-year. Thus, this level of exposure was not associated with any effects.

EPA's comparisons of exposure between the Lockey study and Libby residents are also incorrect. EPA states that the time-weighted exposure for sweeping at the screening plant was 0.25 fibers/ml assuming "thirty minutes" per day, which results in a cumulative exposure of 1.75 fibers/ml-year for a 7-year exposure (the time that the Parkers owned the property prior to the cleanup). We cannot duplicate EPA's calculation. Weis' July 9, 2001 memorandum (Table 2) indicates the personal exposure measurement for sweeping at the screening plant was 0.685 fibers/ml. Assuming a half-hour per day exposure, after this value is multiplied by 0.063 (0.5 hours/8 hours), it gives 0.043 fibers/ml as a time-weighted average workday exposure[5]. For 7 years, the cumulative exposure was 0.3 fibers/ml-year (0.043 fibers/ml times 7 years). The value used by EPA was 6-fold higher. Evidently, EPA made a calculation error. EPA then goes on to compare its incorrect exposure estimate with the exposures from the medium exposure group in Lockey, which is invalid as stated above, as this group did not have elevated lung abnormalities.

In the Lockey study, the group with exposures between 1-10 fibers/ml-year had elevated lung abnormalities that were not statistically significant compared to the controls, but were marginally significant ($P=0.11$). Still, the cumulative exposure for this group was between 3-30 fold higher than for the sweeping scenario at the

[4] The Fisher's exact test is a standard statistical comparison test for calculating the probability value for the relationship between two dichotomous variables.

[5] Typically, an environmental exposure would be based on a 24 hour per day exposure period, which would result in these estimates being reduced 3-fold. However, for the purposes of comparing with the Lockey data, the environmental exposures are based on an 8-hour occupational exposure period.

screening plant. The cumulative exposure in the medium exposure group was about 1 fiber/ml-year, where no effects were observed, which is 3-fold higher than the screening plant exposure. The mean exposure for subjects with pleural changes was 12.1 fibers/ml-yr, which is 40 times higher than the screening plant exposures. The most telling comparison is that the mean cumulative exposure of the "low" exposure subjects in the Lockey study (described as having "limited or no exposure to airborne fibers") was 0.45 fibers/ml-yr, which is higher than the screening plant exposure. Clearly the results from a study where the controls were exposed more than the population in question (pre-remedial exposures at the screening plant) cannot be used to attribute effects to that population.

It is also important to note that the exposure estimate of 0.3 fibers/ml-year at the screening plant is likely an overestimate for several reasons:

- Our analysis of the EPA database showed that the fiber counts for this sample are 3.6 times lower (for all bins) than reported by Weis. If the value in the EPA database is correct, then the exposure estimate for the screening plant needs to be reduced by 3.6-fold (see section III C).
- The assumption of a half-hour of sweeping each day is likely to be a significant overestimate.
- Reanalysis of the sample performed by the RJ Lee Group revealed that the sample appears to have been analyzed improperly (Lee, 2002). In violation of standard procedures, contiguous grid openings were analyzed in portions of the sample, which can bias counting results. Further review showed that the majority of fibers that were counted in this sample were actually cleavage fragments and that some fibers were misclassified as Libby amphiboles (see later discussion showing that cleavage fragments are not toxic). Accounting for these errors, the actual concentration should have been less than 0.1 f/cc, at least 6-fold lower than estimated by EPA.

III. EPA's risk assessment is not transparent or reproducible, and contains numerous apparent errors and inconsistencies. The transparency of the risk assessment does not meet EPA's own guidance on risk assessment or minimum scientific standards. Due to these shortcomings, a complete review of the scientific validity of the risk assessment is impossible. Therefore, EPA's assessments cannot be relied upon, and cannot be supported scientifically.

A. EPA has developed guidelines on the transparency of risk assessments, but the Weis risk assessments did not follow the EPA guidelines for the Libby risk assessments.

Former EPA Administrator Carol Browner introduced the need for transparency and clarity in risk characterization in a March 21, 1995 memorandum to EPA entitled "Memorandum on EPA Risk Characterization Program". In her memorandum, she states that EPA, "must adopt as values transparency in our decision making process and clarity in communication with each other and the public regarding environmental risk ... this means that we must fully, openly, and clearly characterize risks." Along with these statements, an EPA Risk Characterization Policy and Guidance was attached to the memorandum to act as building blocks for the development of policies and procedures, "consistent with the values of transparency, clarity, consistency, and reasonableness." The policy statement developed states that, "A risk characterization should be prepared in a manner that is clear, transparent, reasonable, and consistent with other risk characterizations of similar scope prepared across programs in the Agency."

In December 2000, the Science Policy Council released the Risk Characterization Handbook which implements the March 1995 Risk Characterization Policy. According to the handbook, the "underlying principles for a good risk characterization," are transparency, clarity, consistency and reasonableness (TCCR).

In response to Carol Browner's call for transparency, clarity, consistency and reasonableness in risk characterization the Superfund program has worked to implement these principles by developing Risk Assessment Guidance for Superfund (RAGS): Volume I - Human Health Evaluation Manual (Part D, Standardized Planning, Reporting and Review of Superfund Risk Assessments). Interim guidance was released in 1998, and then followed by final guidance in December 2001, which became mandatory as of June 10, 2002. Planning tools, continuous involvement of EPA risk assessors, and information transfer to a Superfund risk data repository are the key elements of the guidance. Use of RAGS Part D is recommended for removal actions and ongoing risk assessments.

RAGs Part D provides risk assessment table formats for the risk assessor to ensure that all of the risk equation parameters, exposure point data, and calculations are clearly presented. Weis' risk assessments do not provide this level of information. For example, in both the July 9, 2001 and December 20,

2001 memoranda there is no tabulation of the risk estimates; risks are shown as points on small graphs.

EPA has clearly not followed any of this guidance in its risk assessment. In our review of the EPA risk assessments, we have found the following problems that have prevented us from completely understanding the basis for EPA's conclusions:

- Often no calculations, exposure point concentrations, and numerical risk estimates were presented. From my experience, the risk assessments would be unacceptable to EPA if submitted by an outside party.
- We cannot determine which samples EPA used for many of its risk assessment calculations.
- We cannot duplicate many of EPA's calculations.
- EPA based its risk estimates on an index of fiber counts referred to as Phase Contrast Microscopy Equivalents (PCME) (a more complete description of this index is provided in RJ Lee's expert report [Lee 2002]). As discussed in section III C of this report, we cannot determine from the available documentation how EPA estimated the PCME concentration, which is critical to understanding the risk calculations.

EPA acknowledges that the Weis memoranda were lacking in clarity. In a memo from Brattin at SRC to Weis (#495594), Brattin responds to a long series of questions "received from EPA staff" regarding the December 20, 2001, "risk memo." Weis used the Brattin memo to produce his "clarification" memo. However, even with the clarifications provided by Weis, the risk assessment memoranda are still unclear.

B. *EPA developed a complicated database to catalog the asbestos measurement data in Libby, but provided only very limited documentation or training on how to use the database. With any valid risk assessment, all data that are used for exposure calculations should be readily available and clearly documented. However, the disorganized state of EPA's database prevented us from reproducing all of the exposure calculations in the Weis risk assessment. Therefore, the risk assessments do not meet EPA's standards for transparency and reproducibility.*

EPA developed a Microsoft Access database to catalog the asbestos measurement data. This database consists of seven tables which include sampling information, sampling location addresses, results of PLM analyses, results of PCM analyses, results of AHERA analyses and results of ISO10312 analyses. There are over 20,000 records in the sampling information table and over 25,000 records in the analysis tables. No documentation was provided with the database to describe the procedures that were used to create the database or to define the fields that are used in the tables. In some instances, comment fields exist with letter codes in the field but there are no descriptions of what the codes stand for. In a one-hour

training session with EPA representatives and EPA contractors, they were unable to point to any documentation to answer these various questions.

As a result of the inadequacies of the current database, EPA has begun developing a SQL Server database that will replace the Access database. According to EPA, this new database will provide the proper documentation and the missing measurement data. However, at this time, only an incomplete version of the new database has been made available. This inadequacy has prevented a complete evaluation of EPA's findings.

C. *There are numerous apparent errors, inconsistencies, and areas of confusion in the asbestos measurement data used in the risk assessments, which render the risk assessment unreliable.*

The following findings relate to how the asbestos sampling data appears to have been used in the estimation of exposure point concentrations used in the screening level human health risk assessments for Libby. This section focuses on areas where there are apparent inconsistencies and errors between the Libby Asbestos Database and data tables presented by EPA's risk assessor, Chris Weis.

Weis generated three memoranda (dated May 10, 2000[6], July 9, 2001, and December 20, 2001); each memorandum provides risk estimates for various exposure scenarios identified in Libby. The memoranda were intended to support and outline EPA's rationale for determination of imminent and substantial endangerment to public health from asbestos contamination at residential and commercial areas in and around Libby. Weis also wrote an April 24, 2002 memo, which provides further clarification on the earlier risk assessments.

The Database includes results of the Libby asbestos sampling activities. Sampling results are recorded in four separate database tables, one for each of the four analytical methods employed. The analytical methods are described in the RJ Lee report and corresponding Database tables are described below:

International Organization for Standardization (ISO) Method 10312

The ISO 10312 results Database table (tblResultsISO10312) provides detailed information about the results of analyses using method ISO10312. This includes index identification, limited sampling information, sample preparation method, number of grid openings counted, number of fibers counted and concentration of fibers. The number of fibers and fiber concentrations are presented according to size bins and fiber type. The three fiber type categories are Libby Amphibole, Chrysotile, and Other. The size bins were designed to facilitate risk calculations

[6] The copy of this memorandum which forms Attachment 2 of the Action Memorandum (2000) is stamped May 17, 2000. However, this document is later referred to by its author (Weis) as the May 10, 2000 memorandum, that date has been adopted throughout this document.

according to the Berman and Crump methodology, which is not now being used. Further, these bins do not correlate with PCM size requirements.

National Institute for Occupational Safety and Health NIOSH Method 7400 (Phase Contrast Microscopy [PCM]).

The PCM Database table (tblResultsPCM) contains detailed analysis results from the PCM analyses. This table includes index identification, limited sampling information, number of fields counted, number of fibers counted, limit of detection, data qualifier ("<" indicates less than detection limit), and concentration (e.g., fibers/mm^2 of grid and fibers/cc in air).

Asbestos Hazard Emergency Response Act (AHERA)

The AHERA Database table (tblResultsAHERA) contains detailed analysis results from the AHERA analyses. This table includes index identification, area analyzed, number of structures counted (<5μm and >5μm), analytical sensitivity, data qualifier ("<" indicates less than detection limit) and concentration (in structures/mm^2 and structures/cc).

Polarized light microscopy (PLM).

The PLM Database table (tblResultsPLM) contains the results of the PLM analyses. The table includes index identification, data qualifiers and percentage of tremolite-actinolite, chrysotile, other amphiboles, and nonasbestos fibrous material in the samples.

The data presented in Weis' memoranda have been examined in conjunction with the appropriate "snapshot" of the Libby Database.

Weis May 23, 2000 Memorandum / July 2001 Database Snapshot

The first risk evaluation and associated data summary was attached to the Action Memorandum. Asbestos samples collected at the screening and export plants during December 1999 through March 2000 were summarized. The data include 4 indoor air samples for the Screening Plant (SP) and 5 from the Export Plant (EP). A large number (>100) of soil and bulk samples were also taken from these sites. We found the following inconsistencies:

- The EP and SP data are included in the database. However the EP data are not identified as such in the database; the database indicates these as "Not Property Specific."

- The number of soil and bulk material samples listed by Weis for both the SP and EP do not correspond to those found in the database.

July 9, 2001 Memorandum / July 2001 Database snapshot

The July 9, 2001 memorandum presents data summaries for PLM-analyzed soils and bulk materials and PCM/TEM-analyzed air samples targeted for use in certain risk scenarios.

Soils and Bulk-material data

- An error was made in the assignation of non detects for the EP soil and bulk materials (PLM) analysis. Database results with "0" values were counted as detects. It appears that a non-detect was only counted as such if the value was preceded by a less-than sign ("<").

- For all PLM results, the treatment of field duplicates is not specified.

- The number of PLM samples for the SP and schools does not match the data in the Database (note this may be due to insufficient information provided regarding the exact cut off date of Database used by EPA).

PCM results for Screening and Export Plants

- For both EP and SP, the number of PCM-analyzed personal air samples does not match the numbers in the Database.

- The maximum PCM result for the SP is for a sample for which no location is assigned in the Database.

TEM results for Screening and Export Plants

EPA conducted personal air sampling for two EPA workers at each site, one engaged in "sweeping floors" and the other "bagging" activities.

- Both personal air samples analyzed by TEM for EP are indirect preparation. Indirect preparation results in overestimates of the true fiber concentrations. RJ Lee's expert report discusses this issue in depth (Lee, 2002).

- The "bagging" activity analyzed by TEM for SP was by indirect preparation.

- The Database entry for "sweeping floors" at the SP gives a concentration 3.6 times lower than the value in the memorandum (this ratio applies to each of the size bins).

Scenarios 1,2, 3 and Rainy Creek Road

- For many of the PCM and TEM datasets associated with Scenarios 1 through 3 and Rainy Creek Road, the memorandum does not provide sufficient information for us to confirm the number of samples referred to in the memorandum with those found in the Database.

December 20, 2001 Memorandum / December 2001 Database Snapshot and Libby Query Risk Memo 3 Queries

The memorandum authored by Weis of December 20, 2001 provides additional data, revises some of the exposure scenarios described in the July 9, 2001 memo, and develops some new exposure scenarios. As in the other memoranda, the data are presented in the form of summary tables.

Soils and bulk-material data

These media were analyzed by PLM. The data were presented for the following locations categories: yard soil, garden soil, waste piles, driveway soil.

- The results for locations other than the above are not presented in the memorandum. The Database indicates PLM results for the following locations: subsurface soil, track, dirt road, play area soils, and "other". The memorandum does not address or present these results.

- 98 soil samples are assigned subsurface soil designation in the Database's Soil Categories Table despite being listed as surface soils in the Database Field Data table.

- For all categories, the analytical results for field duplicates appear to have been excluded from the summary tables. For samples where more than one analysis was conducted, the highest result was presented. The use of maximum values will introduce a bias, which will result in an overestimation of the concentrations.

Residential Exposure Scenarios

For each scenario, samples were collected using both stationary and personal air samplers ("stationary" and "personal" samples). Airborne fiber concentrations were measured by PCM and TEM for individuals engaged in scenario-specific activities. The results of stationary and personal TEM-analyzed samples are reported by EPA as PCM-equivalent asbestos fiber concentrations (PCME-asb).

- The Database does not appear to support any of the PCME-asb concentrations presented in the memorandum. EPA does not sufficiently explain how the PCME-asb values were derived[7].

- There is no correspondence between the PCME-asb fiber concentrations and information found in the Database table that provides TEM data, the ISO10312 table.

- The ISO10312 table does not provide the individual fiber dimensions for the samples. Prior to database entry, the raw data were grouped into length and size bins; the ISO table only provides the binned data. The bins appear to have been set up to calculate fiber concentrations for use in the Berman and Crump risk methodology. The binned data do not correspond to the size bins necessary to calculate PCME concentrations.

The TEM fiber dimension data are provided in various "source" files (spreadsheets developed by the Volpe Center designed to record the analytical laboratory's raw fiber counts). Raw data files for three samples were compared to the PCME-asb concentration tables generated by EPA's risk assessor. The following samples have non-zero PCME-asb concentrations in Weis' PCME-asb tables, but have no fibers detected according to both the ISO10312 table and the accessible raw data files.

Scenario / Sample Type / Sample Index ID / Volpe Center File ID	Memorandum/risk table asbestos fiber concentration (f/cc)	Raw data file / ISO10312 table asbestos fiber concentration (f/cc)
Scenario 4 (rototilling) / stationary sample / 2-01198 / Volpe PMT162015	0.019	No fibers counted
Scenario 4 (rototilling) / stationary sample / 2-01196 / Volpe PMT161931	0.019	
Scenario 2 (cleaning) / personal sample / 2-01062 / Volpe PMT114967	0.0069	

Another example of inconsistency between the risk assessment and the raw data is shown by two samples used to evaluate Scenario 1 (residential routine activities).

[7] In addition to the core database tables, the Libby Query Risk Memo 3 Queries contains tables generated by EPA's risk assessor. These tables contain the individual sample results that are used to derive exposure point concentrations for use in the risk evaluation. The information in these tables corresponds to the information in the memorandum and with the exception of the PCME-asb table, these "risk" tables agree with the Database. The PCME-asb "risk" table does not reflect the Database information.

Each of these samples underwent four separate TEM analyses. For each sample, two of the analyses were by direct preparation techniques and two by indirect preparation. The raw data files for all indirect preparation analyses (four in total) indicate that fibers were not detected. For each sample, one of the direct preparation analyses returned no fibers detected and the other was a quantified PCME concentration. The table below shows the raw data file information for these samples compared to the values used in the risk memorandum. Even for the higher of the two direct preparation samples (shown in the table), the values are more than order or magnitude lower than the values used by Weis.

Sample Index ID	Memorandum/risk table PCME_mineral concentration (f/cc)	Raw data file PCME concentration (f/cc)[a]
2-00018	0.023	0.00064
2-00022	0.048	0.0017

[a] The highest of the two direct preparation samples

It is important to note that these two samples were the only two Scenario 1 PCME samples with detected fibers. There may well be other examples similar to those described above. It was not possible to exhaustively compare the "risk" values with the raw data due to time constraints and difficulties in extracting the raw data information from the Volpe Center files.

All analytical data from samples with sample filters greater than 25 mm diameter (i.e., the 37 mm filters) are excluded from consideration. The Database contains enough information for the larger filter size samples for a concentration to be computed, but this was not done within the Database. EPA does not explain why the data was excluded. The number of samples that were excluded are summarized below.

Scenario	% of personal air samples excluded due to large filter size	
	PCM	TEM (PCME-asb)
1 (routine activities)	0 %	0 %
2 (cleaning)	25 %	10 %
3 (contact with insulation)	44 %	0 %

The data set used to derive exposure point concentrations contains both direct and indirect preparation sample results. As is discussed in the expert report by RJ Lee (Lee, 2002), it is invalid to include indirect preparation samples. The relative number of indirect preparation analyses is shown below. As indicated in some

cases the maximum value was by indirect preparation. The number of samples with indirect preparation is summarized below.

Scenario	% of indirect preparation analyses in personal air samples used to develop exposure concentrations	
	PCM	TEM (PCME-asb)
1 (routine activities)	2 of 9 = 22%	2 of 5 = 40% (max value is indirect)
2 (cleaning)	6 of 46 = 13% (max value is indirect)	4 of 26 = 15 %
3 (contact with insulation)	1 of 5 = 20 % (max value is indirect)	1 of 5 = 20 % (max value is indirect)

The management of the data used in the risk assessments was inadequate for producing a transparent and reliable risk assessment.

IV. The Weis risk assessments and associated EPA Action Memoranda include unrealistic assumptions and a misunderstanding of key data and studies, which significantly impact the results. These assumptions and misunderstandings tend to overestimate risks.

A. The RJ Lee Group found that EPA inappropriately included cleavage fragments, non-asbestos material, and indirect sample preparations in its fiber counts, which resulted in substantial over counts of fibers.

The RJ Lee Group performed a reanalysis of the EPA measurement data (Lee, 2002). From an analysis of EPA's Access database and the individual count sheets, the RJ Lee Group reported several factors that generated elevated measurement results. First, in violation of the ISO10312 method, indirect preparation was used on numerous samples. In addition, the Quality Assurance Project Plan (QAPP) suggests that indirect preparation samples should only be used in the most limited circumstances, however EPA never discusses this point when presenting risk estimates based on results that include indirectly prepared samples. The sonication that is used in the indirect preparation method breaks apart fiber bundles creating higher measured concentrations (see RJ Lee report for further discussion). Analyses of energy dispersive spectroscopy results indicated that 12% of the spectra were improperly classified as Libby amphibole. Analysis of fiber dimension distribution and sketches of fibers that were counted indicate that 74% of the fibers that were counted were cleavage fragments, not asbestiform particles.

Greater details about the reanalysis of the fiber counts are provided in the expert report by RJ Lee (Lee, 2002).

B. EPA states that the fibers in Libby are of a type and habit that are more toxic than other asbestos fibers. However, analysis of health effects to workers exposed to fibers in the Libby mining operations does not support this claim.

EPA repeatedly states that the fibers in Libby are more potent than other asbestos fibers. Amandus et al. (1987b) and McDonald et al. (1986, 2002) investigated the potency of the Libby fibers in a study of asbestos-related health effects of workers associated with the Libby mining operations. The Amandus and McDonald data were analyzed and it was found that the potency of Libby fibers for lung cancer and mesothelioma is similar to, but generally lower than, the potency factor used by EPA in its risk calculations (Moolgavkar, 2002), which is from EPA's Integrated Risk Information System (IRIS). This analysis demonstrates that the Libby fibers are likely typical, or even less potent than, of other types of asbestos fibers in regards to toxicity and potency.

C. *EPA states that fibers found in the Libby community (i.e., environmental fibers) are as toxic or more toxic than fibers to which Libby workers were exposed (i.e., occupational fibers). However, a comparison of environmental and occupational fibers suggests the opposite.*

The available epidemiologic data on asbestos illnesses are based on studies of occupationally exposed cohorts. However, it is plausible that the fiber dimensions of asbestos in occupational settings may differ from environmental settings, such as in Libby today. Because fiber dimensions are known to effect toxicity (generally long, thin fibers are most toxic), any difference in fiber dimensions between the Libby environmental fibers and those found in occupational settings may influence the appropriateness of EPA risk factors for asbestos.

Weis repeatedly makes the claim that fibers found in the Libby environment today are as toxic or more toxic than fibers found in occupational settings. For example, Weis states in his May 17, 2000, memorandum that "Fibers identified in air include a high proportion of long, thin amphiboles." Unfortunately, in all of this discussion, Weis never provides a comparison of the Libby fibers with fibers from an occupational setting, until his "clarification" memo, and then only with a few samples from Grace in the 1970s.

In a separate report, the RJ Lee Group compared the fiber dimension distributions from EPA samples collected at Libby with the distributions from samples collected in the Amandus study of workers in the Libby mines (Lee, 2002). The details of this analysis are found in the report of RJ Lee. The main conclusion was that Libby environmental fibers are both shorter and wider than the fibers measured in the Amandus occupational study. This suggests that the fibers found in Libby today are less potent than the fibers in Libby mines during the operational periods prior to 1991. Additionally, the Amandus and McDonald cohorts that were exposed to Libby fibers were analyzed, and it was found that the potency of Libby fibers was similar to the potency recommended by EPA and used by Weis in his risk assessment (Moolgavkar, 2002). Therefore, the fiber dimension data suggest that the risk estimates by Weis are not overestimated by using the EPA potency value because (1) the environmental fibers are shorter and wider than the fibers in the mines, and (2) the potency of the Libby fibers that workers were exposed to are similar or lower than the potency estimate used by Weis in the risk assessment.

D. *EPA alleges that its risk assessment is conservative because it did not consider non-cancer endpoints, such as asbestosis. However, asbestosis is generally associated with higher exposures than are currently occurring in Libby.*

My understanding of the body of literature on asbestos health effects is that asbestosis is generally associated with high levels of exposure most often encountered in occupational settings, and would not be expected to occur with the

low environmental exposures in Libby today. For example, in its Airborne Asbestos Health Assessment Update (EPA, 1986a), EPA quotes from a report by the Ontario Royal Commission that concluded "at low levels of occupational exposure to asbestos the fibrotic process in the lungs, if indeed it can be initiated, will not likely progress to the point of clinical manifestation" and further states "the lifetime occupational exposure to asbestos at which the fibrotic process cannot advance to the point of clinical manifestation of asbestosis is the range of 25 f-yr/ml [25 fibers/cc-yr] and below." The post-1990 environmental exposures in Libby are lower than 25 fibers/cc-yr. Based on this comparison, the Weis risk assessment is not likely to be an underestimate of risk.

E. *EPA states that Libby residents are a "sensitive" subpopulation due to prior exposure implying, without evidence, that multiplicative effects will occur with added exposures; rather than a linear, additive effect proportional to the exposure. This statement is inconsistent with EPA's assumptions in its risk assessment.*

EPA states that Libby residents are a "sensitive population" because of historical exposures, particularly for workers associated with the former mine. For this reason, EPA concludes "Asbestos exposures which would present acceptable risks to a healthy population may cause an increase in disease for this highly impacted population." EPA does not provide a basis for these statements.

A simple example can be used to illustrate what EPA is suggesting. Consider a person who is exposed to asbestos for two years in their life, at ages 20 and 50. Further, say the exposure at age 20 results in a contribution to their lifetime risk of 0.001 (1×10^{-3}). At age 50, the person is exposed to a 1000-fold lower contribution that alone contributes 0.000001 (1×10^{-6}) to their lifetime risk. Using conventional risk assessment methods, the total lifetime risk would be 0.001001 (1.001×10^{-3}), the sum of the two risks. In this case, the second exposure added only a very small amount, insignificant to the total lifetime risk. However, EPA is implying that the second exposure would somehow count for more than 1×10^{-6}. This is contrary to conventional risk assessment methods for asbestos, and EPA provides no basis for any mechanism that would support a multiplicative effect.

There is no evidence that prior asbestos exposure leads to increased sensitivity for future asbestos exposures (Hughson, 2002). In addition, EPA implicitly assumes the opposite in its risk calculations. The EPA risk value for asbestos is based on a linear effect (i.e., additional exposures are additive and count the same as prior exposures).

It is true that individuals with prior exposure to asbestos have less future exposure that they can tolerate before disease could occur. However, the additional exposure has to be comparable to the historical exposure to significantly add to the prior exposure. In Libby, there is considerable evidence that historical

exposures were substantially higher than the exposures that exist today. The workers associated with the mine were exposed to concentrations greater than 1 fiber/cc after engineering controls were implemented and as high as 182 fibers/cc before engineering controls were implemented (Amandus et al., 1987a). Also, Weis estimates that the outdoor air concentration in Libby was 1 fiber/cc prior to the closure of the mining operations in 1991 (see Weis addendum of April 24, 2002). Indoor concentrations were also likely higher pre-1991 due to the higher outdoor concentrations. By contrast, the RJ Lee reanalysis found that the exposure point concentrations in indoor environments are considerably lower. EPA has stated that the ambient outdoor concentrations are not a concern (Document #486468 in the administrative record). These data suggest that the current exposures to Libby residents only add small amounts to the cumulative lifetime exposure and related risk.

VI. A substantial portion of the fiber counts from current data collected in Libby were cleavage fragments, not asbestiform fibers. EPA counted the cleavage fragments as having the same toxicity as asbestiform fibers. However, the prevailing scientific opinion is that cleavage fragments are not carcinogenic or toxic. By including cleavage fragments in its fiber counts, EPA substantially overestimated exposures.

A. The scientific weight-of-evidence does not support the conclusion that cleavage fragments are carcinogenic

1. Review of human studies

There has been an opportunity to study the association between cleavage fragments and the incidence of human disease in multiple studies at two study areas: the Homestake Mine in South Dakota and the Reserve Mining Company in Minnesota. In a series of studies published in the peer reviewed literature from the Homestake Mine between 1978 and 1995, no association was found between exposures to the cleavage fragments that result from the cummingtonite-grunerite and tremolite amphibole minerals that are present in the ore that is mined in this study area and the incidence of lung cancer or mesothelioma in the exposed worker population (McDonald et al, 1978; Brown et al., 1986; Steenland and Brown 1995). An earlier study by Gilliam et al. (1976) which examined far fewer subjects (440 compared to 3,328 in the Steenland study) concluded that there was an excess morbidity of malignant and non-malignant respiratory diseases. These early findings by Gilliam in a much smaller cohort have been superseded by the three more recent studies referenced above. The mineral cummingtonite grunerite is a nonasbestiform habit of amosite asbestos. The toxicity of these cleavage fragments is highly relevant to tremolite cleavage fragments.

Further, ore that was mined and used in crushing operations for extraction of iron at the Reserve Mining Company in Minnesota also contains nonasbestiform actinolite and cummingtonite-grunerite. Studies conducted between 1983 and 1992 of workers employed by the Reserve Mining Company did not show an increased incidence of respiratory cancer or any cancer type (Higgins et al. 1983; Cooper et al. 1988 and 1992). The results of studies in these two areas where the predominant exposure was to cleavage fragments is in stark contrast to the high incidence of lung cancer and mesothelioma widely observed in studies where asbestos and asbestiform fibers have been the source of exposure. Therefore, the weight-of-evidence from these human epidemiologic studies involving predominantly cleavage fragments is that the cleavage fragments are not associated with cancer incidence in the exposed worker populations.

2. Review of animal studies

Numerous investigators have explored the carcinogenicity of cleavage fragments and asbestos and asbestiform fibers in animal bioassay studies. These studies have largely employed an injection or implantation procedure that introduces the study materials into the pleural or peritoneal cavity of the test animals. While this method of administration does not reproduce the human experience of inhalation, it does provide an extremely sensitive test for exploring the specific cancer endpoint that is associated with asbestos exposure, mesothelioma. These animal bioassay studies have provided an ample opportunity to explore the carcinogenic potential of cleavage fragments as compared to asbestos and asbestiform fibers. The weight-of-evidence from the studies involving cleavage fragments is that the association between disease incidence in the study animals and the exposures is essentially negative while the studies involving asbestos and asbestiform fibers are highly positive. Below, I summarize these key studies.

Stanton et al. (1981) studied the relation between fiber dimension and cancer of the pleura in rats. Seventy-two particulate samples were examined including asbestos fibers, nonasbestiform amphibole structures and other nonasbestos fibrous materials (e.g., fibrous glass). These materials were implanted in the pleura of rats, and the rats were then observed for 2 years, at which time the survivors were sacrificed. The dose was 40 mg. A high incidence (>90%) of pleural cancer was observed in rats treated with asbestos fibers, whereas the incidence was 0% in rats treated with nonasbestiform tremolite. Based on control data provided in the study, the low incidence observed in some experimental groups was not significantly greater than that in the control group.

Wagner et al. (1982) compared the cancer potential of 3 different types of tremolite (designated Samples A, B and C) in rats. Rats were injected with the samples (20 mg) once into their pleura. Samples A and B contained nonasbestiform tremolite; Sample C was asbestiform tremolite from South Korea. Sample A was injected into 32 Wistar rats two years before the rest of the investigation (experiment 1). Samples B and C were injected into groups of 48 Sprague-Dawley rats. Only Sample C caused mesothelioma cancer (14 of 47 rats). Neither Sample A nor Sample B caused cancer (0 out of 31 rats for Sample A; 0 out of 48 rats for Sample B). It should be noted that there was poor survival of the animals in experiment 2 due to infection. Thus, the incidence of mesothelioma cancer in rats treated with Sample C may have been potentially higher if the animals had survived the treatment period.

Davis et al. (1991) studied the potential cancer effect of 6 forms of tremolite dust samples in rats. The samples were injected into the peritoneal cavity of rats. Three of the samples were asbestiform: California tremolite from Jamestown; Korean tremolite; and tremolite from Swansea. A fourth sample,

Italian tremolite (Ala di Stura), contained cleavage fragments, but it also contained asbestiform tremolite. The remaining two samples were nonasbestiform tremolite from Carr Brae, Dornie, Scotland, and tremolite from Shinness, Scotland. The incidence of mesothelioma cancer was highest for California tremolite (36 of 36 rats), Swansea tremolite (35 of 36 rats) and Korea tremolite (32 of 33 rats). According to Davis et al, the Shinness sample "was almost exclusively composed of cleavage fragments." The Dornie sample "was predominantly made up of cleavage fragments" but "also contained a small proportion of long, thin asbestiform fibers."

The incidences of mesothelioma cancer for rats treated with Italian tremolite were lower (24 of 36 animals). For the Carr Brae and Shinness samples, the incidence of cancer was extremely low (2 out of 36 rats for Shinness tremolite; 4 out of 33 rats for the Carr Brae tremolite). There were no controls in this study, and, therefore, the background incidence rate is not known. However, Pott et al. (1987) showed that intrapleural injection of 1 ml of saline in 32 rats produced 2 mesotheliomas. In addition, Davis et al. commented on the study as follows *"The intraperitoneal injection test is, however, extremely sensitive, and it is usually considered that, with a 10-mg dose, any dust that produces tumors in fewer than 10% of the experimental group is unlikely to show evidence of carcinogenicity following administration by the more natural route of inhalation."* Davis et al concluded that "human exposure to a material such as that obtained from Shinness in Scotland, whether as a pure mineral dust or as a contaminant of other products, will almost certainly produce no hazard, and the material from Dornie is probably to be considered harmless to human beings as well." A more detailed, and confirming evaluation of this background issue concludes that total background is in the range of 15 to 20% (Ilgren and Wagner, 1991).

3. Weight-of-evidence

In conclusion, the overall weight-of-evidence from observations in humans and in animals reveals that exposure to nonasbestiform cleavage fragments does not appear to be associated with respiratory cancers or mesothelioma. According to the U.S. EPA 1986 cancer guidelines, the overall weight-of-evidence for the carcinogenicity of nonasbestiform cleavage fragments would be essentially negative (EPA 1986b). For the sensitive endpoint mesothelioma, the evidence is consistent with Category E - evidence for noncarcinogenicity for humans based on adequate human and animal studies. The human studies included a follow-up analysis of a previously examined human population. The animal studies were conducted in several strains of rats and in hamsters and considered mesothelioma (a relevant cancer endpoint to humans) following pleural or peritoneal injections. Moreover, the results of the human and animal studies demonstrate consistency with regard to the findings regarding cleavage fragments. In contrast, asbestos is classified as a Category A - known human carcinogen. Since this

classification is explicit for fibrous or asbestiform minerals, non-asbestiform minerals are not included. Other classifications include Category B - probable human carcinogen, Category C - possible human carcinogen, and Category D, not classifiable as to human carcinogenicity because of inadequate data. Similar descriptions would be made under the EPA 1999 Draft Cancer Risk Assessment Guidelines, but without a classification label (EPA, 1999).

4. Regulation of asbestos

U.S. regulatory agencies have addressed the biological significance of nonasbestiform fibers, such as tremolite cleavage fragments, and have concluded that these agents do not need to be regulated as a public health hazard. To the contrary, asbestos and asbestiform fibers are regulated as carcinogens. Specifically, in 1992, the Occupational Safety and Health Administration (OSHA) (Fed. Reg. Vol. 57, No. 110, June 1992) deleted nonasbestiform tremolite, anthophyllite, and actinolite from OSHA's asbestos regulations under OSHA's authorities to protect worker health. "Based on the entire rulemaking record before it, OSHA has made a determination that substantial evidence is lacking to conclude that nonasbestiform tremolite, anthophyllite and actinolite present the same type or magnitude of health effect as asbestos. Further, substantial evidence does not support a finding that exposed employees would be at a significant risk because nonasbestiform tremolite, anthophyllite or actinolite was not regulated in the asbestos standards."

The U.S. Environmental Protection Agency (EPA) has a long history of regulating asbestos, but has made a distinction that their rulemaking does not cover nonasbestiform fibers. EPA first issued its Worker Protection Rule in 1986 (EPA, 1986c). The definition of asbestos in the 1986 Worker Protection Rule, and retained in the subsequent 1987 Worker Protection Rule, referred to the asbestiform varieties of the asbestos minerals (EPA 1986c). EPA stated explicitly in the 1987 Worker Protection Rule that it "does not cover nonasbestiform fibers" (EPA 1987).

Likewise, the Consumer Products Safety Commission (CPSC) has considered whether or not to regulate nonasbestiform minerals, in response to concerns regarding the safety of crushed limestone. CPSC did not see evidence of asbestos in limestone and, therefore, determined that regulation was unnecessary. Further, CPSC concluded that the observed tremolite was nonasbestiform cleavage fragments. CPSC evaluated studies and concluded that there is no evidence of a hazard from nonasbestiform tremolite. Quoting the CPSC 1989 decision, the National Toxicology Program (NTP) included the following in its report to Congress on carcinogens: "CPSC in 1989 denied a petition to ban limestone products containing more than 0.1% tremolite because there were no data indicating the presence of asbestiform tremolite in

these products and no data indicating that nonasbestiform tremolite is hazardous" (NTP, 1990).

A number of national and international scientific organizations have evaluated asbestos risk. Scientific evaluations of the health effects of asbestos have been reported by the U.S. EPA in its 1986 asbestos risk assessment document (EPA, 1986a) and are currently in EPA's Integrated Risk Information System (IRIS) database (EPA 1993). These evaluations do not include tremolite cleavage fragments or nonasbestiform substances in the definition of agents associated with the ability to induce cancer. Likewise, the NTP's report on carcinogens which is a document Congressionally mandated to be delivered to Congress on an annual basis, stated in the year 2000 (NTP 2000) that asbestos fibers are carcinogenic. Cleavage fragments or nonasbestiform minerals are not included in this definition of carcinogenic agents. Likewise, the Agency for Toxic Substances and Disease Registry (ATSDR) considers the evidence that inhalation of asbestos leads to increased risk of lung cancer and mesothelioma to be conclusive; nonasbestiform cleavage fragments are not included as carcinogenic in this assessment. Also, the International Agency for Research on Cancer (IARC) has reported that there is sufficient evidence of the carcinogenicity of asbestos; cleavage fragments or nonasbestiform tremolite are not included as carcinogenic in these evaluations (IARC 1987). Finally, in an inter-agency meeting on asbestos held in Denver, Colorado in April 2000, Dr. Vanessa Vu, Associate Director for Health, the EPA National Center for Environmental Assessment (NCEA), and the individual responsible for maintaining EPA's IRIS database, in her presentation stated that "Non-fibrous forms of asbestos are not toxic or carcinogenic." This statement was included as one of several "Asbestos-Related Diseases - General Consensus." The term "Non-fibrous forms of asbestos" includes tremolite cleavage fragments.

In summary, U. S. regulatory agencies, their scientific counterparts, and the IARC have all uniformly determined asbestos and asbestiform fibers are carcinogenic; cleavage fragments and nonasbestiform substances are not considered as carcinogenic agents and are not regulated as posing a health risk.

B. *Anticipating that others would argue that cleavage fragments should not be included in fiber counts, EPA prepared a review (by a contractor) to argue otherwise, but the review is flawed.*

For the Libby site, EPA has assumed that cleavage fragments have the same toxicity as normal asbestiform fibers, which is contrary to common scientific opinion. EPA apparently did not consider the evidence convincing, as Weis predicted that there would be arguments that cleavage fragments are not toxic (document #495593 in administrative record). Further, EPA commissioned an

analysis from Dr. Bill Brattin of Syracuse Research Corporation (SRC) to review this issue (document #495995 in the administrative record).

Dr. Brattin's report cites the study by Davis et al. (1991) in which rats were exposed by intraperitoneal injection to either tremolite asbestos or tremolite cleavage fragments. As Dr. Brattin states in his report, the results of this experiment is open to debate. The one dose of almost pure cleavage fragments produced a 6% mesothelioma rate, a rate which many including one of the authors of the report (Addison) considers to be within the historical control figure for that strain of rat. In his book "Mesothelioma in Animals" (1993), Dr. E. B. Ilgren provides a compilation and analysis of the world's literature on asbestos experiments carried out through 1993. Some of these studies included various types of treated control groups. Dr. Ilgren reports that simply injecting animals intraperitoneally produced mestholioma rates as high as 11.9% and saline alone could produce up to 8% mesotheliomas following intraperitoneal injection.

Dr. Brattin cites three epidemiological studies where exposure to cleavage fragments occurs but where asbestos levels are low or absent. Of these studies, two were negative and are the same studies referenced in this report (see section VI.A.1). I have included a more recent reference to the Minnesota mining study that was overlooked in the Brattin report. The more recent publication by Cooper (1992) provided a third update of the study through 1988 and concluded that the findings are still negative. The Brattin memorandum only cites the initial report by Higgins et al (1983) and concludes that one limitation in the study is the relatively short follow-up time. The third study cited was positive for lung cancers in exposed workers, but as Dr. Brattin points out, there was uncertainty about whether asbestos was present and the confounding influence of smoking was not accounted for. When taken together, these three studies do not, therefore, provide compelling evidence for the toxicity of cleavage fragments; rather the weight of evidence is that cleavage fragments are not associated with cancer incidence in exposed populations.

Considering all the animal and human data available, there is no reasonable scientific basis to support the conclusion that cleavage fragments are toxic.

EPA placed testimony regarding the toxicity of cleavage fragments given to the Senate Committee on Health, Labor, and Pensions in the administrative record (#495603) (the author of the testimony is not clear). The testimony acknowledges a significant difference between asbestos fibers and cleavage fragments. When asked about the tendency of asbestos fibers to fracture longitudinally, producing thinner fibers in the lung, the respondent stated that asbestos minerals have a "tendency to fracture longitudinally," "adding to the fiber lung burden." However, the respondent stated that cleavage fragments have a tendency to "fracture transversely or in an irregular manner."